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Evaluation of the angulation of the nasal septum deviation as an anatomical variation for increased frequency of antral pseudocyst: a cone-beam computed tomography study

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Background: The aim of the study was to determine whether degree of the nasal septum deviation (NSD) can affect the frequency of antral pseudocyst (AP) formation by cone-beam computed tomography (CBCT).

Materials and methods: This retrospective study was included 466 CBCT images. The NSD were categorised into four groups according to the degree: control group (no NSD, 0°–2°), group A (2°–9°), group B (9°–15°), and group C (\geq 15°). The predictor variables were demographic factors (patient's age and gender) and anatomic factors (different degrees of nasal septum angulation). The outcome variable was presence of AP.

Results: Of the 466 cases, 242 (51.9%) had no NSD, 66 (14.2%) had an angle of $2^{\circ}-9^{\circ}$, 111 (23.8%) had an angle of $9^{\circ}-15^{\circ}$, and 47 (10.1%) had an angle of over 15°. The prevalence of AP was 2.04 (95% confidence interval [CI] 1.37 to 3.03; p = 0.001) times higher in the presence of NSD. Significant increases in presence of AP occurred with NSD in group A (2.37 times higher; p = 0.003) and group B (2.07 times higher; p = 0.003) compared to control by univariate analysis.

Conclusions: Although there is no sufficient evidence to suggest that NSD is a definitive aetiological factor for AP development, our findings indicated that NSD increased the risk of AP formation. (Folia Morphol 2023; 82, 1: 158–165)

Key words: nasal septum, deviation, antral pseudocyst

INTRODUCTION

Antral pseudocysts (APs) are radiopaque, domeshaped lesions that do not contain a true epithelial lining and are composed of a serous exudate. APs are predominantly located in the maxillary bone and associated with the Schneiderian membrane [37]. These pathological entities are asymptomatic and can be detected during radiological examination particularly by plain radiography and/or cone-beam computed tomography (CBCT). APs are considered to be associated with allergic reactions, humidity, temperature, trauma, smoking, odontogenic infection, although its pathogenesis remains controversial [7, 8, 26, 29]. In the sinus elevation procedures through crestal or lateral approach, AP can also be caused by the perforation, trauma and infection of

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the Schneiderian membrane. AP might be unilateral or bilateral in the maxillary bone and the dimension of AP might increase gradually, remain the same, or decrease gradually [16, 38, 39]. AP cannot disrupt the integrity of the cortical plate of the maxillary sinus and of the Schneiderian membrane [16, 39].

Nasal airflow directly affects the development of craniomaxillofacial bones and paranasal sinuses [22]. Obliteration of the nasal passage caused by nasal septum deviation (NSD) prevents optimal nasal breathing and also results in mouth breathing [4]. Nasal septum is defined as a midline structure of the nasal cavity which divides the nasal cavity into halves and supports the external nose structurally [13]. Nasal septum consists of cartilaginous (anteriorly, quadrangular cartilage), bony (posteriorly, vomer and perpendicular plate of the ethmoid bone) and membranous components [13]. NSD is an anatomic variation that affects approximately 80% of adults [36, 43]. In the literature, there are various classifications of NSDs [5, 19]. Cerkes [9] defined five main nasal septum deviation, named as caudal septal deviation, anteroposterior C-shaped deviation, cephalocaudal C-shaped deviation, anteroposterior S-shaped deviation and cephalocaudal S-shaped deviation. Guyuron et al. [19] reported that the most frequent type of NSD was characterised by an inclined septum.

Maxillary sinus pneumatisation and airway obstruction in the nose are predisposing factors for pathologies in the maxillary sinus [35]. The maxillary sinus volume might change according to the capacity of the nasal airflow [15, 30]. NSD, concha bullosa, and ostium obstruction might alter the pneumatisation of the maxillary sinus, thereby leading to the thickening of the maxillary sinus membrane and chronic sinusitis [23]. NSD is also an aetiological factor affecting the nasal airway and pneumatisation of the maxillary sinus [23].

Although possible aetiological factors in the development of AP have been reported in the literature, no study evaluating the relationship between NSD and AP development has been identified. We aimed to investigate whether the presence and degree of NSD has an effect on the development of AP by using CBCT.

MATERIALS AND METHODS

Study sample

Initially, seven hundred CBCT scans obtained between January 2016 and July 2018 and retrieved

from the archives of the Faculty of Dentistry, Izmir Katip Çelebi University were included. CBCT scan were taken for orthodontic treatment, oral and maxillofacial examination and dental implant treatment. The following inclusion criteria were applied: patients older than 18 years, nasal area and both maxillary sinuses entirely visible on the scan with AP present or absent. Exclusion criteria included inadequate scan quality, impacted teeth in the maxillary sinus, nasal and antral sinus infection, cyst and tumour associated with maxillary sinus and/or nasal structure, pseudocyst related to odontogenic infection, age under 18 years, S-shaped septum deviation, multifaceted nasal septum deviation, concha bullosa, previous maxillofacial and/or paranasal surgery, and maxillofacial trauma. After detailed examination, a total of 466 patients with CBCT scan (236 male, 230 female) met our inclusion criteria.

The study was approved by the Ethics Committee of Izmir Katip Çelebi University. (Approval No.: 230), and the study protocol was conducted in accordance with the Helsinki Declaration 1975 as revised in 2000.

CBCT image acquisition, evaluation of the antral pseudocyst and nasal septum

All the images were obtained using a CBCT device (NewTom 5G CBCT machine, QR s.r.l., Verona, Italy). The exposure parameters were as follows: 110 kVp, 1–20 mA, with 15 × 12 cm field of view and resolution mode (voxel size 0.2 mm). The maxillary CBCT scans were obtained from the level of the maxillary teeth crown to the top of the orbit. All CBCT images were evaluated by the same experienced surgeons (NL and MY) on a desktop UltraSharp LED TFT 24-inch monitor (Dell Inc., Round Rock, TX, USA) with appropriate background lighting.

Antral pseudocysts were defined as unilacunar, dome-shaped, radiopaque lesions with no bone resorption on the wall of the maxillary sinus, particularly on the floor of the sinus (Fig. 1) [1, 8, 17, 20, 24, 25, 28, 39, 44]. AP location was classified as right, left and bilateral based on the location of the lesions in the maxillary sinus.

The location of the concavity was determined based on the direction of the deviation [2]. The NSD angle was measured between the two lines drawn on the coronal section of CBCT with the first line drawn from the crista galli to the anterior nasal spine and the second line drawn from the crista galli to the most prominent point of the deviation



Figure 1. Radiographic appearance of antral pseudocysts in the maxillary sinus.

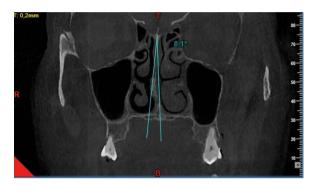


Figure 2. The nasal septum deviation angle was measured between the two lines drawn on the coronal section of cone-beam computed tomography, with the first line drawn from the crista galli to the anterior nasal spine and the second line drawn from the crista galli to the most prominent point of the deviation.

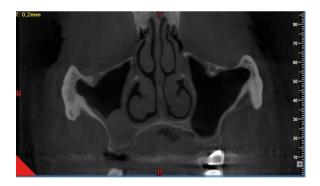


Figure 3. Radiographic appearance of straight nasal septum.

(Fig. 2) [2]. Patients were divided into four groups based on the degree of NSD: i) control group (no NSD, $0^{\circ} < \text{NSD} \le 2^{\circ}$), ii) group A (mild, $2^{\circ} < \text{NSD} \le 9^{\circ}$), iii) group B (moderate, $9^{\circ} < \text{NSD} \le 15^{\circ}$), and iv) group C (severe, NSD $\ge 15^{\circ}$) [43]. Directions of NSD were divided into three groups: i) straight nasal septum (control group, $0^{\circ} < \text{NSD} \le 2^{\circ}$), ii) nasal septum deviated to the right, and iii) nasal septum deviated to the left. These measurements were



Figure 4. Radiographic appearance of the nasal septum deviated to the right.

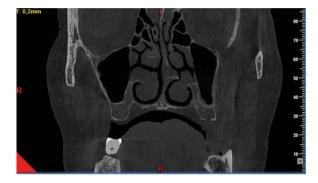


Figure 5. Radiographic appearance of the nasal septum deviated to the left

made in the coronal view and are demonstrated in Figures 3, 4 and 5.

Statistical analysis

Statistical analyses were performed using SPSS for Windows version 22.0 (IBM Corp., Armonk, NY, USA). Binary and multinomial logistic regression analyses were used to estimate the odds ratio (OR) and 95% confidence interval (CI). Gender and age were included in the models to adjust their effects on the dependent variable. Descriptive statistics were expressed as frequencies (n), percentages (%), and mean ± standard deviation (SD). Intra-operator reliability for each measurement was assessed using the intra-class correlation coefficient (ICC), with an interval of at least 3 weeks between measurements, which yielded a 98% agreement rate. The predictor variables were demographic factors (patient's age and gender) and anatomic factors (different degrees of nasal septum angulation). The outcome variable was presence of AP. The relation between predictors and outcome variable was analysed using binary and multinomial logistic regression analyses, with statistical significance set at a p value less than 0.05.

Table 1. Data of the mean age and nasal septum deviation (NSD) angle

	Number of patients	Minimum	Maximum	Mean	Standard deviation
Age	466	18	83	40.53	18.170
NSD angle	466	.0	33.9	5.819	6.9304

Table 2. Descriptive statistics for 224 cases with nasal septum deviation (NSD)

	Number	Minimum	Maximum	Mean	Standard deviation
NSD angle	224	4.6	33.9	12.106	4.8689

Table 3. Distribution of cases according to the gender, antral pseudocysts (AP) and nasal septum deviation (NSD)

		N	%
Gender	Male	236	50.6
	Female	230	49.4
AP in maxillary sinus	Absence	316	67.8
	Right	55	11.8
	Left	48	10.3
	Bilateral	47	10.1
NSD angle	Absence	242	51.9
	$2^{\circ} < \text{NSD} < 9^{\circ}$	66	14.2
	$9^{\circ} \leq NSD < 15^{\circ}$	111	23.8
	$NSD \geq 15^{\circ}$	47	10.1

RESULTS

According to the ICC test, the concordance index was greater than 0.98, indicating high intra-examiner concordance.

The 466 patients included 50.6% (n = 236) men and 49.4% (n = 230) women with a mean age of 40.53 ± 18.17 (range: 13–83) years and mean NSD angle 5.81 ± 6.93 (Table 1). A straight nasal septum (control group) was detected in 242 (51.9%) images and NSD was detected in 224 (48.1%) images (mean NSD angle, 12.10 ± 4.86) as shown in Table 2. Of the 224 NSD cases, 66 (14.2%) cases had an NSD angle of 2° –9°, 111 (23.8%) of them had an angle of 9° –15°, and 47 (10.1%) of them had an angle of over 15° (Table 3). APs were detected in 150 (32.2%) cases, among whom the lesion was localised on the right side in 55 (11.8%), on the left side in 48 (10.3%), and on both sides in 47 (10.1%) cases (Table 3). Distribution of the AP was shown in Table 4.

Binary logistic regression analysis indicated that the prevalence of AP was 1.82 (95% CI; range: 1.23–2.7) times higher in men than in women (p = 0.003). Moreover, the prevalence of AP was 2.04 (range:

1.37-3.03) times higher in the presence of NSD than in the absence of NSD (p = 0.001) and the age- and gender-adjusted OR was 1.97 (95% CI: 1.32-2.93; p = 0.001). The prevalence of AP was 2.54 (95% CI: 1.60-4.03; p = 0.001) times higher in the right maxillary sinus with NSD compared to the control group and the age- and gender-adjusted OR was 2.44 (95% CI: 1.52-3.89; p = 0.001). However, no significant difference was found between the prevalence of AP in the left maxillary sinus and in the control group (p = 0.069). On the other hand, the prevalence of AP was 2.37 times higher in group A and 2.07 times higher in group B compared to the control group and the age- and gender-adjusted results were statistically significant as well (p = 0.003 and p = 0.005, respectively). Nevertheless, no significant difference was found between group C and the control group (p = 0.190) (Table 5).

DISCUSSION

Aetiological factors of pathological entities or anatomical variations in the maxillary sinus typically include upper respiratory tract infection, trauma, congenital deformities, odontogenic infection/cyst//tumour, adenotonsillar hypertrophy, and chronic nasal airway obstruction [3, 11]. Some of these entities might be symptomatic such as acute sinusitis, odontogenic cyst while some of them might be asymptomatic such as mucocele, pseudocyst, antral polyps, and mucous retention cyst. The pathologies in the maxillary sinus are generally detected incidentally in routine radiographic and/or clinical examination.

The CBCT enables a three-dimensional examination for the assessment of the anatomic structure and pathologies of the maxillofacial region, thereby allowing a definitive diagnosis. Three-dimensional CBCT images were used in the current study for the detection of the NSD type, measurement of the NSD

Table 4. Distribution of the antral pseudocysts according to the nasal septum deviation (NSD), gender, direction of the NSD and NSD angle

	Location of antral pseudocysts							P-value	
	Right		Left		Bilateral		Absence		_
	N	%	N	%	N	%	N	%	_
NSD									0.05
Absence	23	41.8	18	37.5	19	40.4	182	57.6	
Presence	32	58.2	30	62.5	28	59.6	134	42.4	
Gender									0.007
Male	36	65.5	24	50	31	66	145	45.9	
Female	19	34.5	24	50	16	34	171	54.1	
Direction of NSD									0.009
Straight	23	41.8	18	37.5	19	40.4	183	57.9	
Right	18	32.7	19	39.6	18	38.3	66	20.9	
Left	14	25.5	11	22.9	10	21.3	67	21.2	
NSD angle									0.011
Absence	23	41.8	18	37.5	19	40.4	182	57.6	
$2^{\circ} < \text{NSD} < 9^{\circ}$	13	23.6	7	14.6	9	19.1	37	11.7	
$9^{\circ} \leq NSD < 15^{\circ}$	13	23.6	20	41.7	12	25.5	66	20.9	
$NSD \geq 15^{\circ}$	6	10.9	3	6.3	7	14.9	31	9.8	

Table 5. Multivariate analysis of factors associated with antral pseudocysts

Variables		Antral ps	eudocysts					
	Presence (n = 150)		Absence (n = 316)		Univariate		Age and gender adjusted results	
	Mear	± SD	Mean	± SD	OR [95% CI]	Р	OR [95% CI]	Р
Age	42.14 ± 16.04		39.77 ± 19.08		1.01 [0.97–1.02]	0.188		
	N	%	N	%	_			
Gender					_			
Male	91	60.7	145	45.9	1.82 [1.23–2.7]	0.003*		
Female	59	39.3	171	54.1	1 (reference)			
Direction of NSD								
Straight	60	40.0	183	57.9	1 (reference)		1 (reference)	
Right	55	36.7	66	20.9	2.54 [1.60-4.03]	0.001*	2.44 [1.52–3.89]	0.001*
Left	35	23.3	67	21.2	1.59 [0.96–2.63]	0.069	1.56 [0.94–2.59]	0.084
NSD								
Presence	90	60	134	42.4	2.04 [1.37–3.03]	0.001*	1.97 [1.32–2.93]	0.001*
Absence	60	40	182	57.6	1 (reference)		1 (reference)	
NSD angle								
Absence	60	40.0	182	57.6	1 (reference)		1 (reference)	
$2^{\circ} < \text{NSD} < 9^{\circ}$	29	19.3	37	11.7	2.37 [1.35–4.19]	0.003*	2.39 [1.35–4.26]	0.003*
$9^{\circ} \leq NSD < 15^{\circ}$	45	30.0	66	20.9	2.07 [1.28–3.37]	0.003*	2.01 [1.24–3.25]	0.005*
$NSD \geq 15^{\circ}$	16	10.7	31	9.8	1.57 [0.80–3.06]	0.190	1.41 [0.71–2.78]	0.324

^{*}Significant at the 0.05 level; Binary logistic regression model; CI — confidence interval; NSD — nasal septum deviation; OR — odds ratio; SD — standard deviation

angle, detection of AP, and the evaluation of the maxillofacial structure.

Previous studies indicated that the nasal obstruction and impaired intranasal airflow caused by NSD

have an adverse effect on mucociliary activity and the resulting ciliary loss has been shown to increase the incidence of rhinosinusitis [14, 31, 32, 42]. However, although the relationship between NSD and chronic diseases of the sinus has been documented, an exact positive correlation has not yet been established due to inconsistent findings presented by the studies. In a 2009 study, Polat and Dostbil [34] evaluated patients with NSD using rhinoscintigraphy and reported that the administration of septoplasty improved the reduced nasal mucociliary transport rate. Although there have been several studies supporting this finding, Greguric et al. [18] and Collet et al. [10] reported that they found no definitive role of NSD in the pathogenesis of chronic rhinosinusitis [31]. Similarly, Yasan et al. [46] revealed that the mild-to-moderate septal deviation was not a risk factor for chronic sinusitis and only severe septal deviation was found to be a risk factor for the disease. In a confirmatory manner, a systematic review conducted in 2010 found a significant relationship between a septal deviation of > 10° and rhinosinusitis [31]. Although the studies abovementioned have investigated rhinosinusitis, the present study examined the possible effect of NSD on AP associated with inflammatory factors and a comparison was only made with the studies abovementioned since there are a limited number of studies reporting on the relationship between NSD and AP.

In our study, 90 (60%) of the patients were detected with NSD and Binary logistic regression analysis indicated that the prevalence of AP was 2.04 (range: 1.37-3.03) times higher in patients with NSD than in patients without NSD (p = 0.001). Additionally, the prevalence of AP was 2.37 (range: 1.35-4.19) times higher in group A ($2^{\circ}-9^{\circ}$) and 2.07 (range: 1.28–3.37) times higher in group B (9°-15°) compared to the control group $(0^{\circ}-2^{\circ})$ (p = 0.003 for both). However, although no significant difference was found between group C ($\geq 15^{\circ}$) and the control group with regard to AP prevalence (p = 0.190), this prevalence was 1.41 (range: 0.71-2.78) times higher in group C compared to the control group. These findings are rather consistent with the results of the study by Orlandi [31] who found a significant relationship between a septal deviation of >10° and rhinosinusitis.

On the other hand, rhinosinusitis, particularly chronic rhinosinusitis, is considered to be associated with allergies, bacteria, viruses, biofilms, osteitis, superantigens, immunological dysregulation, and gastroesophageal reflux [6, 27]. It has also been re-

ported that a single independent factor could not be held responsible and that there could be multiple factors leading to the development of rhinosinusitis [6, 27]. Additionally, although NSD is known to cause ostiomeatal complex narrowing and thereby to aggravate inflammation, the contralateral inflammation in NSD has been reported to be associated with different aetiologies [33]. Perloff et al. [33] suggested that the inflammation spread to the contralateral side through the Haversian system and another hypothesis posited that contralateral inflammation was caused by the nasal secretions pushed into the sinus during nose blowing. Some other studies indicated that the increased airflow on the side contralateral to the deviated septum and the turbulence in the medial meatus lead to ciliary loss in the maxillary sinus and also increase inflammation on both sides [21, 40]. Moreover, NSD has also been radiologically shown to be associated with the presence of contralateral concha bullosa [14, 41].

Antral pseudocysts have been reported to be associated with environmental factors including allergic reactions, smoking, trauma, changes in the air temperature, and humidity [7, 8, 26, 29]. A recent study by de Carvalho et al. [12] reported a significant relationship between the presence of AP and greater height of the maxillary sinus ostium. Another study that was conducted by the two authors of the present study and awaits publication revealed that AP could be associated with the masticatory forces transmitted to the sinus membrane through the protruded healthy maxillary molar roots penetrated into the maxillary sinus [45].

Given that NSD alone could not be held responsible for the development of rhinosinusitis, we consider that NSD alone could not be held responsible for the development of AP as well. This hypothesis could be supported by our finding that indicated that AP was detected in patients with NSD and on the non-deviated (unaffected) side as well, considering that it would be more reasonable to expect that the risk of inflammation on the deviated side would be relatively higher [31]. Accordingly, based on the inflammatory effect of NSD in the pathogenesis of rhinosinusitis reported in the literature, it could be asserted that NSD could have an effective role in the aetiopathogenesis of AP.

Our study was limited in several ways. First, the study only included the clinical symptoms of NSD and the patients with right- or left-side deviation due to its nature. Accordingly, it did not include the other NSD types and thus included no evaluation on the effect of other NSD types. Secondly, no evaluation was performed on the relationship between AP and the pathologies that could be associated with nasal airway obstruction other than NSD.

CONCLUSIONS

Although there is no sufficient evidence to suggest that NSD is a definitive aetiological factor for AP development, our findings indicated that NSD increased the risk of AP formation.

Conflict of interest: None declared

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